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Retrospective Investigation of Alcohol Intoxications Followed in Intensive Care Unit

Yoğun Bakımda Takip Edilen Alkol Zehirlenmelerinin Retrospektif İncelenmesi

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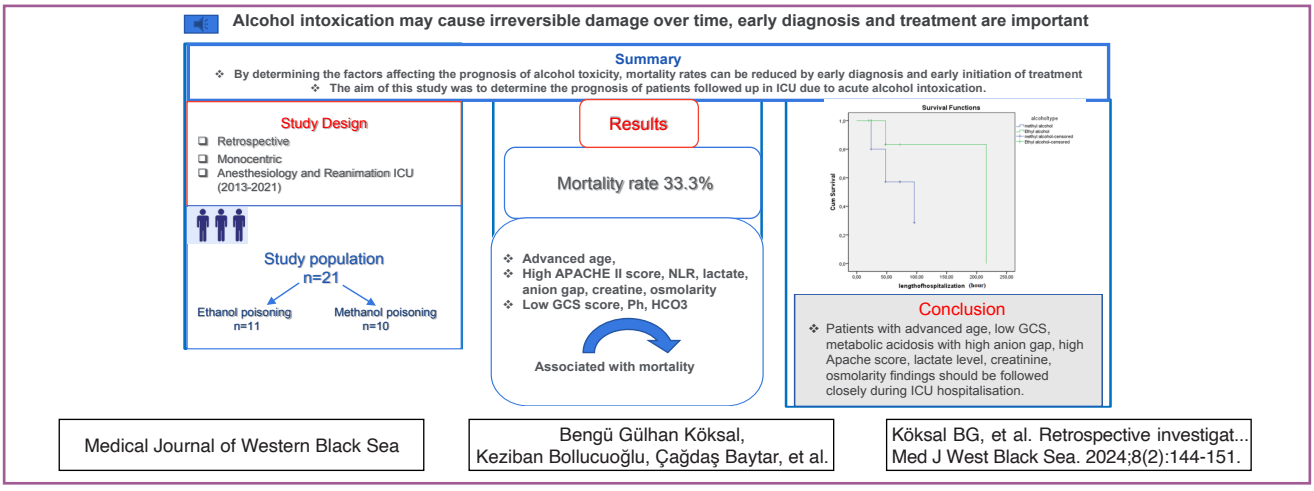
GRAPHICAL ABSTRACT



ABSTRACT

Aim: The majority of alcohol-related deaths are due to acute alcohol consumption. There are many factors affecting the prognosis of alcohol toxicity. It has been reported that by determining these factors, mortality rates can be reduced by early diagnosis and early initiation of treatment. In this study, we aimed to determine the prognosis by evaluating the clinical status and laboratory factors of patients followed up in intensive care unit (ICU) due to acute alcohol intoxication.

Material and Methods: The study included 21 patients with acute alcohol intoxication who were followed up in the ICU of our hospital between 2013-2021. Laboratory parameters, demographic characteristics and clinical status of the patients were recorded. Patients were divided into both exitus and survivors and according to the type of alcohol consumed (ethanol and methanol).

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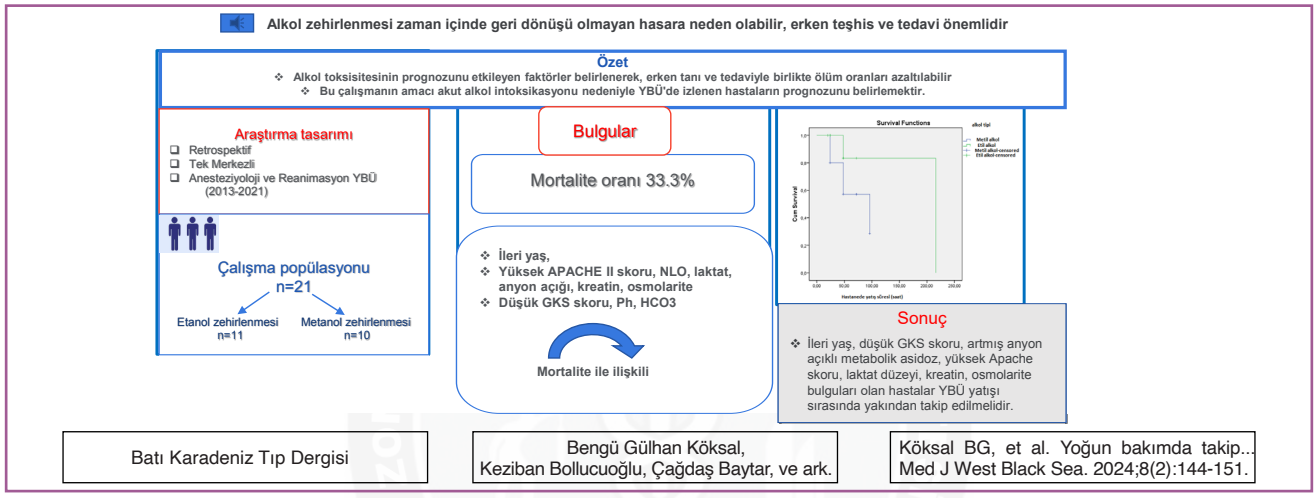
Results: All patients were male with a mean age of 40.10 ± 15.9 years. 52.4% (n=11) of the admissions were ethanol poisoning and 47.6%(n=10) were methanol poisoning. Mortality rate was 33.3%. It was observed that symptoms appeared later in methanol intoxication compared to ethanol intoxication (p<0.001). When ethanol and methanol groups were compared, pH and HCO3- levels were lower and lactate and creatinine levels were higher in Group M. Advanced age, low Glasgow coma scale (GCS) and high Acute Physiology and Chronic Health Evaluation (APACHE II) scores, high neutrophil/lymphocyte ratio (NLR), lactate, anion gap, creatinine, osmolarity and low pH, HCO3- were associated with mortality (p<0.05).

Conclusion: In this study, advanced age, low GCS, high APACHE scores, metabolic acidosis with high anion gap in laboratory findings, increased NLR, lactate level and creatine elevation were found to be associated with mortality in acute alcohol intoxication. We think that patients with these findings should be followed closely.

Keywords: Ethanol, methanol, intoxication, mortality

GRAFİKSEL ÖZET



ÖZ

Amaç: Alkolle ilişkili ölümlerin büyük bir kısmı akut alkol tüketiminden kaynaklanmaktadır. Alkol toksisitesinin prognozunu etkileyen birçok faktör vardır. Bu faktörlerin belirlenmesi ile erken tanı konulup tedaviye erken başlanarak mortalite oranlarının azaltılabileceği bildirilmiştir. Bu çalışmada akut alkol zehirlenmesi nedeni ile yoğun bakımda takip edilen hastaların klinik durumu ve laboratuvar faktörleri değerlendirilerek prognozun belirlenmesi amaçlanmıştır.

Gereç ve Yöntemler: Çalışmaya 2013-2021 yılları arasında hastanemiz yoğun bakım ünitesine (YBÜ) takip edilen ve akut alkol intoksikasyonu tanısı alan 21 hasta dahil edildi. Hastaların laboratuvar parametreleri, demografik özellikleri ve klinik durumları kaydedildi. Hastalar hem eksitus olanlar ve hayatta kalanlar olmak üzere hem de tüketilen alkol tipine (etanol ve metanol) göre gruplara ayrıldı.

Bulgular: Olguların tümü erkek hastalardan oluşuyordu ve yaş ortalaması 40.10 ± 15.9 yıl olarak tespit edildi. Başvuruların %52.4'ü ethanol, %47.6'sı metanol zehirlenmesiydi. Mortalite oranı %33.3 tü. Metanol zehirlenmesinde semptomların, etanol zehirlenmesine kıyasla geç dönemde ortaya çıktığı gözlendi (p<0.001). Etanol ve metanol grupları karşılaştırıldığında Grup M'de pH, HCO3- düzeyleri daha düşük, laktat ve kreatinin değerleri ise daha yüksek bulundu. İleri yaş, yüksek Akut Fizyoloji ve Kronik Sağlık Değerlendirmesi (APACHE II) skoru , nötrofil/lenfosit oranı (NLO), laktat, anyon açığı, kreatin, osmolarite ve düşük Glaskow koma skalası (GKS) , pH, HCO3- mortalite ile ilişkiliydi (p<0.05).

Sonuç: Bu çalışmada, akut alkol zehirlenmesinde ileri yaş, düşük GKS ve yüksek APACHE skoru, laboratuvar bulgularında yüksek anyon açığı ile seyreden metabolik asidoz, artmış NLO, laktat düzeyi ve kreatin yüksekliği mortalite ile ilişkili bulunmuştur. YBÜ yatışı sırasında bu bulgulara sahip hastaların yakından takip edilmesi gerektiğini düşünüyoruz.

Anahtar Sözcükler: Etanol, metanol, intoksikasyon, mortalite

INTRODUCTION

Ethanol and methanol play important roles as precursors and/or solvents in chemical synthesis. Ethanol is also used in pharmacology and food industry for drug dissolution (1). Alcohol intoxication refers to a harmful condition caused by the accumulation of alcohol and its metabolites in the bloodstream faster than they can be metabolised by the liver, caused by recent alcohol intake. The main clinically remarkable side effects of alcohol are neurological, gastrointestinal and cardiovascular problems which are generally related with the alcohol concentration in the blood (2,3) When the blood alcohol level reaches 200-299 mg %, symptoms of intoxication occur except in individuals with high tolerance. When the blood alcohol level reaches 300 mg%, coma and death may result in young individuals and individuals with new alcohol use experience. (4). Between 2010 and 2012 in the United States of America, acute poisoning was found to be the direct cause of an average of 2,221 deaths in the sample group aged 15 years and older. The same route of death is thought to indirectly cause more than 30,000 deaths per year (5). The prognosis of alcohol toxicity depends on many factors, including chronicity of use, degree of intoxication, associated traumatic injuries and end-organ damage (6).

The aim of this study was to determine the prognosis of methanol and ethanol intoxication by evaluating clinical status and laboratory factors in patients followed up in intensive care unit. The secondary aim was to reveal the differences between ethanol and methanol alcohol poisoning.

MATERIAL and METHODS

Study Population

After obtaining faculty ethics committee (ethical approval No. 2020/22-18), 21 patients who were followed up with the diagnosis of acute ethanol and methanol intoxication in the Anesthesiology and Reanimation Intensive Care Unit (ICU) of Zonguldak Bulent Ecevit University Hospital between January 2013 and March 2021 were included in the study. Patients with trauma, haematological disease, infection, immunosuppressive drug use and patients with missing clinical and laboratory data were excluded. The diagnosis of ethanol intoxication was based on blood ethanol level. The diagnosis of methyl alcohol intoxication was based on clinical suspicion and supportive diagnostic tests since the patient's blood methanol level could not be determined.

Data Collection

Medical records, laboratory and radiologic findings and complaints at admission were retrospectively evaluated by accessing archival data and information processing automation system.

Demographic data of the patients (age, gender), type of alcohol causing intoxication, duration of ICU stay, discharge status, complaints at admission, Glasgow Coma Scale (GCS) score, ocular findings, and computed tomography results, Inotropic support and mechanical ventilation requirements, Acute Physiology and Chronic Health Evaluation (APACHE) II score, laboratory findings (hemogram, biochemistry, blood gas values, osmolarity, anion gap, osmolar gap), and medical treatment information were recorded. Among the hemogram parameters; white blood cell, hemoglobin, hematocrit, lymphocyte and neutrophil values were evaluated and Neutrophil/Lymphocyte ratios were calculated. The anion gap was calculated from the laboratory test as follow: $(Na^+ + K^+) - (Cl^- + HCO3^-)$. The osmolar gap was determined by subtracting the measured serum osmolality from the calculated serum osmolality (2(Na) + glucose/18 + BUN/2.8). Patients were divided into two groups as survivors and non-survivors and according to alcohol type as ethanol (Group E) and methanol (Group M) intoxication.

Statistical Analysis

SPSS 22 program was used for data analysis. Descriptive statistics were calculated as frequency and percentage for categorical variables, mean ± standard deviation and median (minimum-maximum) for continuous variables. Mann-Whitney U test was used to compare quantitative and ordinal data and Pearson's chi-squared test and Fisher's exact test was used was used for qualitative data to evaluate the relationship between the outcome of alcohol intoxication and influencing factors. In all analyses, $p<0.05$ was accepted as significant level.

RESULTS

Twenty-one patients who were followed up in the ICU of our hospital with the diagnosis of alcohol intoxication were included in the study. The mean age of these patients was 40.10 ± 15.9 years. All of the patients were male. 52.4% (n=11) of the admissions were ethanol intoxication and 47.6% (n=10) were methanol intoxication. In Group E, the mean blood alcohol levels were 170±79.41. Three patients who developed coma due to ethyl alcohol intoxication had blood alcohol levels of 366 mg/dl, 252 mg/dl and 217 mg/dl. One patient had a blood alcohol level of ≥300 mg/dl although he was not in a coma. There was a history of chronic alcohol use in 85.7% of the cases. The mean time to onset of symptoms in acute poisoning cases was 7.33±6.35 hours. In methanol intoxication, symptoms appeared statistically significantly later than in ethanol intoxication (12.30±5.77, 2.81±1.88, $p<0.001$, respectively). The median length of stay in the ICU was 48 (20-216) hours.

The most common symptoms in patients admitted to the ICU were loss of consciousness (52.4%), visual loss (28.6%), and nausea and vomiting (14.3%). One patient (4.3%) was admitted to the ICU after cardiopulmonary arrest. All patients with visual complaints were admitted to the ICU due to methanol intoxication.

In Group M, one patient received symptomatic treatment, one patient received hemodialysis+fomepizole, and the other patients received hemodialysis+ethanol+ folate treatment. In patients with ethanol intoxication, hemodialysis was performed in 2 patients and symptomatic treatment was performed in the other patients. It was determined that 9 patients in Group M and 1 patient in Group E needed hemodialysis.

Radiologic findings were present only in Group M and in 50% of these patients. Three patients had putaminal necrosis, one patient had subarachnoid hemorrhage and one patient had both.

The mortality rate due to alcohol intoxication was 33.3%. In the analysis, advanced age, high APACHE II score, neutrophil/lymphocyte ratio (NLR), lactate, anion gap, creatine, osmolarity and low GCS score, Ph, $HCO3^-$ were associated with mortality. The need for mechanical ventilation and inotropic support increased in patients with mortality (respectively p=0.001, p<0.001) (Table 1,2).

Table 1: Association of clinical and demographic characteristics of patients with mortality.

	Survivor Group (n=14)	Exitus Group (n= 7)	p
Age, year	32 (18-57)	53 (33-69)	0.007*
Time of onset of symptoms (hour)	3.5 (1-20)	10 (2-24)	0.172*
GCS score	14 (4-15)	3 (3-10)	<0.001*
APACHE score	7.5 (1-16)	28 (14-46)	<0.001*
Length of stay in hospital (hour)	48 (20-96)	48 (24-216)	0.488*
Visual disturbances Yes/No, n (%)	3 (21.4%)/ 11 (78.6%)	3 (42.9%) / 4 (57.1%)	0.354**
Alcohol type ethanol/methanol	9 (64.3%) / 5 (35.7%)	2 (28.6%) / 5 (71.4%)	0.183***
Need for inotropic support Yes/No, n (%)	0 /14 (100%)	6 (85.7%) / 1 (14.3%)	<0.001**
Need for hemodialysis Yes/No, n (%)	4 (28.6%) / 10 (71.4%)	4 (28.6%) / 10 (71.4%)	0.159**
Need of mechanical ventilation Yes/No, n (%)	1 (7.1%) / 13 (92.9%)	6 (85.7%) / 1(14.3%)	0.001**

*Mann Whitney U, ** Fisher's exact test, ***Pearson chi square test. Test Values are presented as mean ± standard deviation, number (%) or median (minumum-maximum) , **GCS:** Glasgow coma scale, **APACHE:** Acute Physiology and Chronic Health Evaluation

Table 2: Association of patients' laboratory values with mortality.

Parameters	Survivor Group (n=14)	Exitus Group (n= 7)	p*
pH	7.36 (7.23-7.44)	7.06 (6.74-7.31)	<0.001
PaO_2 (mmHg)	95 (61.4-161)	144 (56-263)	0.057
$PaCO_2$ (mmHg)	39.5 (12-50)	44 (12-51)	0.197
$HCO3^-$ (mmol/L)	22.1 (10.8-26.1)	9.8 (5.3-21.1)	0.006
Lactate	1.7 (0.8-8.9)	4.4 (2.2-16)	0.002
Osmolarity (mOsm/L)	290 (268-293.3)	292 (290-319)	0.038
Anion gap (mEq/L)	9.25 (4-24.2)	35.7 (9.1-42)	0.002
Osmolar gap (mOsm/L)	10 (3.56-24.5)	15 (2.68-30.42)	0.172
Glucose (mg/dL)	135.5 (92-249)	123 (69-200)	0.636
WBC	10.2 (6.2-14.3)	14.1 (3-28.1)	0.149
NLR	3.55 (0.85-7.1)	8.76 (1.92-22.8)	<0.001
Hb (g/dL)	15 (11.6-17.2)	13.6 (11.2-16.5)	0.056
Htc (%)	43.6 (37.5-52.2)	41.5 (32.9-49.2)	0.494
Urea (mg/dL)	20 (14-57)	31 (21-61)	0.128
Creatine (mg/dL)	0.9 (0.5-1.2)	1.6 (1.2-3)	0.005
ALT (U/L)	13 (8-70)	40 (12-198)	0.067
AST (U/L)	20.5 (13-209)	84 (20-766)	0.052

*Mann-Whitney U test , Values are presented as mean ± standard deviation, **WBC:** White blood cell count, **NLR:** Neutrophil to Lymphocyte Ratio, **Hb:** Hemoglobin, **Htc:** hematocrit **ALT:** Alanine transaminase, **AST:** Aspartate aminotransferase

Table 3: Comparison of laboratory values of deaths according to Methanol and Ethanol poisoning.

Parameters	Group M (n=10)	Group E (n= 11)	*p**
pH	7.17 (6.74-7.44)	7.37 (7.27-7.41)	**0.004**
PaO_2 (mmHg)	126.5 (61.4-169)	95 (56-263)	0.314
$PaCO_2$ (mmHg)	40.65 (12-50)	40 (31.5-51)	0.756
$HCO3^-$ (mmol/L)	10.8 (5.3-24.8)	22.3 (17.8-26.1)	**0.001**
Lactate	5.75 (0.8-16)	1.8 (1.1-4.4)	**0.020**
Osmolarity (mOsm/L)	291 (275-314)	290.7 (268-319)	0.809
Anion gap (mEq/L)	26.7 (12-41.9)	8 (3.9-14.9)	**<0.001**
Osmolar gap (mOsm/L)	15.1 (2.68-30.42)	10 (3.56-24.4)	0.152
Glucose (mg/dL)	149.5 (69-249)	105 (92-211)	0.557
WBC	12.1 (7.5-28.1)	10.2 (2.9-27.1)	0.314
NLR	4.97 (0.85-16.42)	3.55 (1.12-22.8)	0.426
Hb (g/dL)	13.8 (12.5-17.2)	14.7 (11.2-16.5)	0.468
Htc (%)	42.1 (39-52.2)	43.6 (32.9-49.2)	0.710
Urea (mg/dL)	31.5 (14-61)	20 (15-32)	**0.024**
Creatine (mg/dL)	1.2 (0.6-3)	0.9 (0.5-2.4)	**0.034**
ALT (U/L)	13.5 (11-198)	15 (8-59)	0.972
AST (U/L)	34 (16-766)	27 (13-209)	0.597

*Mann-Whitney U test , Values are presented as mean ± standard deviation , **WBC:** White blood cell count, **NLR:** Neutrophil to Lymphocyte Ratio, **Hb:** Haemoglobin , **Htc:** hematocrit **ALT:** Alanine transaminase , **AST:** Aspartate aminotransferase

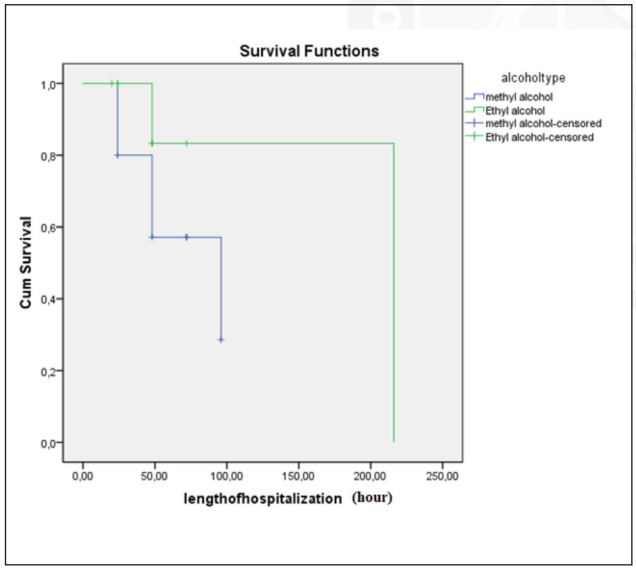


Figure 1: Kaplan-Meier survival curve of patients with alcohol intoxication in intensive care unit.

When ethanol and methanol groups were compared, lower pH, $HCO3^-$ levels and higher lactate and creatinine values were found in Group M (p=0.004, p=0.001, p=0.02, p=0.034, respectively.) (Table 3).

The Kaplan-Meier survival curve obtained in all patients is shown in Figure 1.

DISCUSSION

In this study, the mortality rate from alcohol intoxication was 33.3%. Advanced age, high Apache score, NLR, lactate, anion gap, osmolarity, creatine and low pH, $HCO3^-$, GCS were found to be associated with mortality. The need for mechanical ventilation and inotropic support increased in mortal patients. When ethanol and methanol alcohol groups were compared, lower pH,$HCO3^-$ levels, higher lactate and creatinine values were found in methyl alcohol poisoning.

A study conducted by Morteza Bagi et al. revealed a mortality rate of 3.7% among individuals presenting to the emergency department with alcohol poisoning, with all of these cases attributed to methanol poisoning (7). The mortality rate for methanol poisoning was estimated to be 20%. Similarly, a study conducted by Büberci et al. reported a mortality rate of 38.9% in cases of methanol poisoning (8). In our study, the overall mortality rate was 33.3% in all cases of alcohol poisoning. However, this rate increased to 50% in cases of methanol poisoning and decreased to 18.2% in cases of ethyl alcohol poisoning. It is believed that these variations could be attributed to differences in the sample size and the timing of hospital admissions.

Alcohol intoxication can lead to severe metabolic acidosis characterized by high anion and/or osmolar gaps, along with the potential for acute renal failure in certain patients. In cases of methanol intoxication, the initial metabolic path-

way involves conversion to formaldehyde and subsequently to formic acid. The early-stage acidosis primarily results from the accumulation of formic acid. Formic acid, in turn, inhibits mitochondrial cytochrome-c oxidase, leading to increased anaerobic respiration and lactate production. Additionally, low-molecular-weight ethanol can contribute to an osmolar gap by elevating the measured serum osmolality above the calculated osmolality (9-13). A study investigating the correlation between demographic characteristics, physical examination findings, laboratory results, and the survival of patients with methanol poisoning in the emergency department revealed significant differences. Specifically, creatinine and base deficit levels were notably higher in the survivor group compared to the non-survivors, while bicarbonate and pH levels were significantly lower (14). In their study examining alcohol poisoning and its outcomes, Morteza Bagi et. al. found a significant association between an increase in potassium, creatinine, blood sugar, dialysis requirement, and hematocrit levels with patient mortality. Furthermore, an increase in dialysis requirement and creatinine level was calculated as independent risk factors for mortality (7). The results of our study demonstrated lower pH and bicarbonate levels, alongside higher lactate and creatinine levels in cases resulting in death. The comparison between the ethanol and methanol groups revealed more pronounced metabolic acidosis in the methanol group, with lactate levels being approximately three times higher.

The sensitivity and specificity of anion gap and osmolar gap in diagnosing toxic alcohol poisoning are limited. The presence of a normal osmolar gap does not rule out the possibility of toxic alcohol poisoning. Any osmotically active substance in the blood can elevate serum osmolality above normal levels. When the osmolar deficit is within the range of ≤15 – 20 mOsm/L, the suspicion of toxic alcohol accumulation arises. It has been suggested that an osmolar deficit exceeding 25 mOsm/L can be a useful indicator to initiate treatment (15). In the early stages of methanol poisoning, the osmolar gap typically exceeds 20 mOsm/kg H2O. However, in the later stages, the osmolar gap may return to normal as toxic formate concentrations increase during methanol metabolism (16). Although the mean osmolar gap was higher in non-survivors in our study, there was no significant difference between the two groups. Besides that, the serum osmolarity was significantly higher in non-survivors. There was no significant difference in osmolar gap and serum osmolarity between cases of methyl and ethyl alcohol poisoning. This absence of difference may be attributed to the inclusion of some patients who presented at advanced stages, where the osmolar gap may become normal. Since the diagnosis of anion gap toxic alcohol poisoning lacks sensitivity, patients may exhibit symptoms of toxic alcohol ingestion shortly after consumption without presenting with anion gap metabolic acidosis. As the anion gap increases, the osmolar gap may decrease, making toxic alcohol poisoning more likely, although it does not serve as a definitive diagnostic indicator (17). Our study demonstrated significantly higher anion gap in non-survivors compared to survivors and in cases of methanol poisoning compared to ethanol poisoning.

Acute kidney injury represents a life-threatening complication in cases of poisoning. The primary causes of acute kidney injury (AKI) include ischemia, hypoxia, or nephrotoxicity (18). It was reported that acute kidney injury developed in a patient with a methanol concentration of 400 mg/dL during admission, along with myoglobinuria (19). Chang et al. reported that AKI occurred in 66% of cases following methanol poisoning, and it was associated with a 19.67 times higher risk of in-hospital mortality (20). Although the specific etiology of AKI could not be determined in our patients, creatine levels were notably elevated in non-survivors and in cases of methyl alcohol poisoning.

In their study, Stankevic et al. identified that acute alcohol intoxication induced alterations in various inflammation-related markers linked to alcohol metabolism and hepatocellular damage. They also noted differences in marker responses to excessive alcohol consumption in individuals with alcohol-related liver disease, non-alcoholic fatty liver disease, and healthy controls (21). Massey VL and Arteel G reported that acute alcohol exposure increased the susceptibility to infection during acute poisoning, potentially exacerbating liver damage due to the inflammatory response to such infections (22). NLR is a widely recognized indicator calculated using neutrophil and lymphocyte values from complete blood count, typically serving as an indicator of subclinical inflammation (23). NLR exhibits its role as an acute stress marker earlier than other laboratory parameters, with an early increase observed within less than six hours following acute physiological stress (<6 hours). NLR has demonstrated predictive value for poor outcomes in various diseases. It has been demonstrated that in uncomplicated cirrhosis, a high NLR can predict mortality independently of the Model for End-Stage Liver Disease (MELD) and Child-Pugh scores. In this study, the mean NLR values were 2.08±0.99 for survivors and 4.39±3.0 for non-survivors. Notably, the survival rate of patients with an NLR of at least 2.72 was significantly lower (24,25). While there remains no consensus on the normal NLR values across different age groups and genders, Forget et al. established that normal NLR values in a healthy adult population typically fall within the range of 0.78 to 3.53 (26). In our study, NLR was significantly higher in non-survivors compared to survivors (10±6.34 and 3.51±2.12, respectively). While the comparison of the methyl and ethyl alcohol groups no significant difference, both groups exhibited relatively high mean NLR values (6.09 ±4.60, 5.46 ±6.23). We are of the opinion that NLR can serve as a valuable prognostic indicator in cases of alcohol poisoning; however, it should be considered in conjunction with other relevant variables.

The Glasgow Coma Scale score is widely employed as an indicator of mortality following various neurological events. A GCS score of less than 8 points typically signifies severe deterioration. Recent studies have indicated that the GCS may also hold relevance for assessing impaired consciousness stemming from intoxication (27). A study that examined 844 patients with ethanol poisoning admitted to the emergency department revealed that 57% of the patients exhibited only mild impairment of consciousness (GCS ≥13), 20% displayed moderate impairment (GCS 9-12), and 23% had severe impairment (GCS ≤8). Notably, all patients with a high degree of impaired consciousness recovered without complications, and none required mechanical ventilation (28). A multicenter study suggested that one of the most potent predictors of poor outcomes after methanol intoxication was the presence of coma (GCS score <8) (29). Lee et al. further demonstrated that the GCS score served as a significant risk factor associated with mortality in cases of methanol poisoning. In our study, the median GCS score among patients who did not survive was 3 (3-10) (30). Moreover, patients presenting with methanol poisoning exhibited significantly lower GCS score compared to those presenting with ethanol poisoning.

In a cohort study, it was noted that the APACHE II score, utilized as an early warning indicator for mortality, proves to be a valuable tool for clinical prediction of hospital mortality. It was highlighted that none of the patients with APACHE II scores of 31-40 or 21-30 survived (31). In our study, the median APACHE II score among patients with a fatal outcome was 28 (14-46).

In cases of alcohol poisoning, symptoms are typically correlated with the blood alcohol content (BAC). In cases with blood alcohol content exceeding 300 mg/dL the risk of respiratory depression and arrest is escalated. Fatalities due to acute alcohol intoxication are most commonly observed when the BAC exceeds 500 mg/dL, although the lethal dose of alcohol can vary (3). Among the patients monitored for ethanol intoxication, the mean blood alcohol content was 170±79.41 mg/dL. Remarkably, three patients who developed coma due to ethyl alcohol poisoning had blood alcohol levels of 366 mg/dL, 252 mg/dL, and 217 mg/dL upon arrival at the hospital. One patient experienced only nausea and vomiting, despite having a BAC of ≥300 mg/dL. This difference may be attributed to factors such as the quantity of alcohol consumed, individual body weight, alcohol tolerance, the alcohol concentration in the beverage, and the duration of alcohol consumption. The amount of methanol that leads to toxicity depends on the solution's concentration and metabolic processes. In adults, toxicity has been documented with as little as 15-500 ml of a 40% solution. Diagnosis confirmation relies on a positive serum methanol or formate assay. However, current techniques, such as gas or liquid chromatography, can be laborious, expensive, and often unavailable (32). Thus, a thorough understanding of the clinical presentations and significant laboratory features of methanol poisoning patients is vital for diagnosis. Since methanol levels of the patients included in our study could not be measured, a diagnosis of methanol poisoning was established based on patient history, clinical examination, and laboratory results.

In methanol poisoning, toxic effects of methanol in the nervous system can be seen on CT and MR imaging. Putaminal necrosis with or without hemorrhage and subcortical white matter lesions are the most commonly reported findings (33). In our study, radiologic findings were present in 50% of the patients with methanol intoxication in accordance with the literature. Three patients had putaminal necrosis, one patient had subarachnoid hemorrhage and one patient had both.

The study is subject to certain limitations. Firstly, it was designed as a retrospective study. Secondly, the sample size was relatively small. Thirdly, it was not possible to measure the methanol levels in the patients. Due to the small sample size, definitive cut-off values could not be established in our study.

In conclusion, since alcohol intoxication may cause irreversible damage over time, early diagnosis and treatment are important. Advanced age, low GCS, metabolic acidosis with high anion gap (low pH, low HCO3 level), high Apache score, lactate level, creatine, osmolarity in laboratory findings may be signs of mortality. Therefore, we think that patients with these findings should be followed closely during ICU admission.

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None.

Author Contributions

Concept: **Bengü Gülhan Köksal, Keziban Bollucuoğlu,** Design: **Bengü Gülhan Köksal, Gamze Küçükosman,** Data Collection or Processing: **Bengü Gülhan Köksal, Çağdaş Baytar,** Analysis or Interpretation: **Bengü Gülhan Köksal, Çağdaş Baytar,** Literature search: **Rahşan Dilek Okyay, Bengü Gülhan Köksal,** Writing **Bengü Gülhan Köksal, Özcan Pişkin, Hilal Ayoğlu,** Approval: **Hilal Ayoğlu.**

Conflicts of Interest

All authors declare no conflict of interest.

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Ethical Approval

The study was approved by the Clinical Resarches Ethics Committee of Zonguldak Bülent Ecevit University (Date: 18/11/2020 and 07.04.2021, No:2020/22-18).

Review Process

Extremely and externally peer-reviewed and accepted.

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